CEREBRAL CIRCULATION PREVAILING DURING SLEEP AND HYPNOSIS

BY J. WALLACE NYGARD

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The General Problem

This study is a comparison of the cerebral circulation prevailing in natural sleep with that prevailing in hypnosis, as well as an investigation of the adequacy of the so-called 'cerebral circulation theories' of sleep. These have been of two opposing types: one group espousing cerebral anæmia as the cause of sleep, and another the cerebral congestion view.

Mosso (13) in 1881 from observations on three trephined patients during sleep concluded that sleep is associated with an increased volume of blood in the limbs as a result of relaxed vessels and a coincident decreased supply to the brain. Howell (8) continued the plethysmograph study of the extremities during sleep and, since he found the volume of the periphery to increase during sleep, he assumed that there must necessarily be a reduced volume in passive brain vessels. From these experiments, Howell postulated the cerebral anaemia theory of sleep. This assumes that the sympathetic

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center becomes fatigued from continued activity during the day, and, as a result of this fatigue, those blood vessels of the body supplied with vasomotor nerves relax. The relaxation results in an increase of volume and a reduction in blood pressure in the periphery which, in turn, is sufficient to produce a diminution in volume of the passive brain vessels (cerebral anæmia), causing sleep. Such a theory seemed very plausible. Bayliss and Hill (1), Roy and Sherrington (14), Hill (7), and other investigators at that time envisaged the cerebral vessels as being without vasomotor fibers and therefore regulated entirely indirectly by circulation changes in other parts of the body. If the blood volume could be shown to increase in the extremities during sleep, and the blood pressure to decrease, what could be more natural than to believe that there would be a diminution in the intracranial circulation?

Studies by Brush and Fayerweather (4), Blackenhorn and Campbell (2), Landis (11), Brooks and Carrol (3), and others have clearly shown that the blood pressure does decrease in sleep, and their data supports Howell's viewpoint. However, the assumption that the cerebral vessels act only passively to circulatory changes elsewhere in the body is one that recent investigators have rejected in the light of newer experimental studies. It is now conceded that the cerebral vessels have their own vasomotor nerve supply and may vary independently of the circulation changes elsewhere in the body. Numerous studies have shown this indirectly but it remained for Forbes and Wolff (5) in microscopic photography of the pial vessels through an implanted skull window in the dog to give the most direct evidence for such a view. Other workers corroborated the findings of Forbes and Wolff, and with other technical advances have revealed without a doubt that brain vessels actively change in size, and that these vessels may vary independently of the general blood pressure.[1] Moreover, even Mosso's original experimental data from which Howell's theory derives its strength have been contra-

[1] For an excellent review of the literature of this subject, the reader is referred to Wolff, H. G., The cerebral circulation, *Physiol. Rev.*, 1935, 16, p. 545.

dicted by more recent experimental work done by Shepard (15). In an extensive study of two trephined cases, he has shown that, contrary to the views held by Mosso, sleep is characterized by a long and sustained increase in volume. Similar results were found by Stevenson, Christianson and Wortis (16) on several brain hernia cases. However, the latter authors, as a result of their study, developed a cerebral congestion theory of sleep which we will later have reason to criticize. They envisaged the sympathetic center in the brain as becoming periodically fatigued with a resulting vaso-dilatation of both brain and periphery. This vaso-dilatation might cause sleep in either of two ways: "the brain volume would be increased with a consequent pulling apart of the neurons (diaschisis)"; or "the cerebrospinal fluid would be increased in pressure within the brain, especially in the pericullular spaces, thus altering the conductivity of the synapse."

Gibbs (6) has developed a technique in which brain circulation in individuals with intact skulls can be studied. By a thermo-flow recording needle he measures the blood flow as it leaves the brain through the internal jugular vein. Gibbs found no difference in circulation between the waking and sleeping states.

Previous to this investigation there seem to have been no attempts made to observe the cerebral circulation prevailing during hypnosis. The volume of blood in the arm during hypnosis, as determined by a plethysmograph, has been compared to that during sleep by Walden (19). He found that the circulation changes during hypnosis were, in general, the reverse of those found in sleep, which led him to suggest that the cerebral circulation may also be different. However, other workers (17, 18) have not found the prolonged gradual constriction in the periphery that Walden found so characteristic of hypnosis. Numerous investigations, in fact, have shown that the resemblance between hypnotic sleep and normal sleep is only superficial. Hull has pointed out in his book that actually hypnotic sleep resembles the waking condition more than it resembles natural sleep. Since the appearance of Hull's book, Jenness and Wible (19), in a well controlled

study comparing respiration and heart action in sleep and hypnosis, report cardiac rate and respiration as lowered in sleep but not in hypnosis. Similarly, Loomis, Harvey, and Hobart (12) in measuring brain potential waves during induced hypnosis report that the waves in the trance condition resemble those in the waking condition rather than in sleep.

Subjects

The subjects used in this study were three young males and one female. Each of these individuals had had a portion of the skull removed, making possible measurements of cerebral circulation changes.

Subject No. 1 (female) had been admitted to the University Hospital on February 19, 1936 with a skull fracture and right extra-cerebral hemorrhage as a result of a toboggan accident. A decompression and right temporal craniatomy was performed. The extra-cerebral hemorrhage was evacuated, and a small portion of the cortex, which was softened, was also removed. Observable slight Jacksonian convulsions were alleviated by operation. Recovery was uneventful. She attended college during the school year of 1936–1937 with no observable effects of the injury and experiments were run with her from January to May, 1937. The bone defect was in the shape of an ellipse, measuring about 6.5 cm in its longest and 4.5 cm in its shortest diameter. Pulsations in the region of the defect were easily noticeable. The scalp was loose and free and the hair could be parted away from the defect so that shaving the head was unnecessary.

Subject No. 2 (male) had been admitted to the University Hospital with a compound comminuted fracture of the frontal bone, as a result of an automobile accident. The brain was oozing out of the lacerations. On November 17, 1935 a debridemant skull fracture and partial craniectomy were performed. A part of the frontal lobe of the brain was removed and the frontal sinuses excised. Both olfactory nerves were destroyed. Since release from the hospital four convulsions have been reported but none during the school year 1936–1937 when we carried on our observations. Experiments with this subject ran from November, 1936 through May, 1937. The injury had healed well. No plate was used. The defect in the skull measured about 7.5 cm by 2.5 cm and had relatively even edges. The skin overlying the defect had a considerable dip, but was loose and free and could be observed noticeably to pulsate with each heart beat.

Subject No. 3 was an epileptic boy with a left temporal decompression. We include several observations during normal sleep in this subject, but actually they were incidental observations obtained while making a study of intra-cranial pressure in epilepsy.

Subject No. 4 (male) had a compound skull fracture as a result of an automobile accident. A right subtemporal decompression with the dura incised was done at Johns Hopkins Hospital, October 30, 1930. Recovery was uneventful and complete. The experiments on this subject were conducted in the physiology department of Johns Hopkins University during the summer.

Experimental Rooms and Apparatus

Two adjoining rooms, not interconnected but both opening into a large laboratory, were used in this investigation. In one room were set up the recording apparatus,

piston recorders, Marey tambour (recording device for pneumograph), two signal magnets, syringes, and special blood pressure apparatus. The other room was the sleeping room with bed and necessary bed clothes. Between the sleeping room and apparatus room there were holes in the wall through which thick walled rubber tubing connected the recording devices with the brain tambour system, pneumograph, and scalp control attached to the subject. The bed and the subject were in semi-darkness with only enough light entering through the open door so that experimenter could observe the sleeper. The experimenter usually sat in the doorway at the head of the bed. Nearby was placed paper, pencil, and signal key to record movements of the sleeper on the kymograph paper. An assistant supervised the apparatus, but, once the writing levels were recording, the apparatus needed very little attention, so that the assistant usually watched the recording while the experimenter was free to observe the sleeping subject. The temperature of the room was recorded during each experiment at various periods during the night, but, since it seldom varied more than one degree during the night, the effects of temperature changes are negligible. It was necessary, however, to wait 15 minutes after the subject was placed in the apparatus to allow for any possible temperature changes in the tambour system to attain a normal level.

To measure the volume of the brain in subject 2 a plaster of paris cast of the forehead was taken, and from it was constructed a tambour that curved with the forehead and rested comfortably inverted on the bone adjacent to the depression. A thin dam of rubber was stretched over the surface, so that when the tambour was placed over the defect, the tension of the rubber dam held the skin down in the defect of the skull, but would move with each heart beat and any changes in volume. Some of the experiments were conducted with a piece of balsa wood attached to the rubber dam to press the scalp still further down in the defect. The tambour system was sealed in place by plastic modeling clay and held in position firmly by a broad bandage wrapped around the head from front to back. For each of the subjects 1, 3 and 4 a tambour was constructed to rest comfortably inverted on the bone adjacent to the defect and attached to the rubber dam of the tambour by means of beeswax was a piece of balsa wood cut to fit the depression. The tambour was held firmly in position by one broad bandage wrapped around the head from front to back. Additional tambours were constructed for two of the subjects to act as scalp controls. In subject 2 the scalp control was placed above the cranial defect tambour on the forehead; on subject 1 on the opposite side from the cranial defect. These served in detecting changes due to movement as well as changes in the vascular bed of the scalp.

The electrically driven kymograph in the apparatus room had wide drums and could carry a nine foot paper. Sufficient smoked papers were always at hand so that a night's work could continue without interruption. All brain tracings and some scalp controls were written by a piston recorder lubricated by distilled water; this was of standard make, having a bore of 1.6 cm and a celluloid writing level of 14.5 cm in length.

Respiration was measured from the thorax by a Sumner pneumograph attached to a Marey tambour.

Numerous other studies have shown that the blood pressure falls with the onset of sleep. We checked this observation with subject 2. We preferred to use the usual ausculation method to determine systolic and diastolic pressure. Readings were taken on the right arm before and at irregular periods during sleep.

In addition to these irregular readings, we also set up a continuous method of measuring blood pressure that would record changes in blood pressure that appear as waves, synchronously with respiration and brain volume changes. To obtain these

continuous records of blood pressure, a modified Erlanger capsule and mercury manometer was set up in the apparatus room. The pneumatic bag on the left arm of the subject in the sleeping room was inflated from the recording room slightly above diastolic pressure as previously determined on the other arm by the ausculation method. A suitable Marey tambour attached to the modified Erlanger capsule recorded these changes synchronously with respiration and cranial defect changes on the smoked drum. It should be made clear that this latter method does not record quantitative measurements but does show general changes in pressure, especially the respiratory waves of blood pressure. It was not possible to keep the pneumatic bag continually inflated during the act of going to sleep, but we were able to obtain records while awake and during periods of as much as eight minutes duration while asleep without awakening the subject.

Procedure

Most of the records were obtained from the subjects on the left side position. Subject 2 also preferred a side position in sleeping, but since the cranial tambour, in his case, recorded better with the subject lying on his back, he was required to sleep in this position.

The procedure in all sleep observations was very simple. After the apparatus was attached and due allowance made for temperature adjustment, the lights were turned out and the kymograph started. Our subjects did not have any difficulty in falling asleep rather quickly. After a suitable period of sleep, the subject was aroused. To have corresponding data on the awake condition we at times gave the command, "wake up!" when the subject was actually awake.

Minor variations in general procedure were introduced on certain evenings to answer such questions as:

1. Do changes in respiration have any influence on the volume curve?
2. Do the circulatory changes precede sleep or are they the result of sleep?
3. Is the blood pressure reduced in sleep as reported by so many authors?
4. Does the increase in the vascular bed of the scalp by tightening of the bandage give a simulation of increased volume in the cranial defect tambour?

The cerebral circulation prevailing during hypnosis was not studied until sufficient data of sleep had been obtained. An attempt was made to induce a deep hypnosis in all subjects, but it was successful only in two subjects, namely, subjects 1 and 4. With suitable suggestions these subjects appeared to be in a profound somnambulistic hypnotic trance, with catalepsy demonstrated by supposedly involuntary retention of the arm in position where placed, and an apparent amnesia post-hypnotically for all trance events and suggestions. All the experimental sessions on hypnosis were held at the same hours in the evening as the sleep experiments.

Before any experiments were conducted upon induced hypnosis, the subject was trained until only a few words sufficed to induce the trance. Post-hypnotic amnesia, local analgesia, post-hypnotic suggestions, hallucinations, and catalepsy were demonstrated in these training series. The procedure used in actual experimental observations corresponded with that of the study of sleep. A brief record was taken while awake and then hypnosis was induced. To insure the subject was not in normal sleep or awake, the subject was tested at intervals by catalepsy (retention of the arm in position where placed).

We differed from the usual procedure on two occasions in each subject by inducing the hypnosis before the apparatus was attached. This variation was introduced so

that other tests than catalepsy could be used to measure the depth of hypnosis obtained. On some evenings the interval testing of hypnosis was dispensed with, on the assumption that testing for hypnosis might act as a suggestion for hypnosis, and thus keep the trance condition from merging into sleep. On these evenings only one test instead of tests at approximately 15 or 20 minute intervals followed the hypnosis suggestion. In subject 4 interval testing of suggested analgesia was also introduced to insure that hypnosis had not merged into sleep.

Results of Sleep Observations

We have a total of thirty-four experiments on four subjects in going to sleep, during sleep, at awakening, and after awakening. In addition, there will be discussed some minor experiments that were designed to answer certain questions that arose.

All brain tracings were written by a piston recorder having a bore of 1.6 cm in diameter, with a writing lever of 14.5 cm from tip to axis. The distance from the axis to the insertion of the piston rod was 3.2 cm. The actual movement of the piston would therefore be given approximately by dividing the movement of the writing point by 4.5. Due to the large bore, it was obviously not delicate enough to record changes in the vascular bed of the scalp but readily recorded the pulsations from the region of the defect, since these were very marked. However, to test whether the dilatation of the vascular bed could, by tightening the bandage, produce a simulation of increased volume in the region of the defect, it was necessary to test with a control tambour placed over a region of intact skull. In subject 1 the control tambour was identical with the tambour overlying the defect except that the balsa wood was cut flat instead of rounded to fit the defect. The control tambours, when used, were placed on the forehead directly above the cranial defect tambour in the case of subject 2, and on the opposite temporal bone from the defect in the other subjects. Although the scalp controls were written by a piston recorder of smaller bore and therefore more delicate than the piston recording brain volume, no increase in volume during sleep was apparent from these controls. They did serve a very useful purpose, however, in recording artificial changes due to movement. Notes were taken by the observer of all movements of the sleeping sub-

ject. An electric indicator controlled by a signal key marked the point on the smoked drum where the notes were taken. It became a simple matter to detect artificial changes in the brain volume curve with the scalp control. Movements always produced a sharp rise and fall unaccompanied by the usual pulse form changes. True vasomotor changes were always less abrupt with full pulse form showing at all times. The largest source of movement was not change of position in sleep, but the stretching of strained muscles after being awakened from a period of sound sleep. These always appeared in the volume curve as a sharp break. In spite of being shown the necessity for doing so, subjects had difficulty in controlling the desire to stretch after being roused.

Lengthy records were taken of the subjects awake at rest, but nowhere were considerable volume changes observable. The pulse form awake is characterized by a pulse low in amplitude, with a low dicrotic notch at, or very near the base of the wave. (See last portion of Fig. 1, and first portion of Fig. 2 of Plate I.)

When sleep ensues the amplitude of the arterial pulse increases markedly, and the dicrotic wave becomes elevated and much more prominent. With these pulse changes the volume of the blood in the brain increases. The pronounced pulse and increased volume are probably due to relaxed vessels. The rise in volume is gradual and usually rhythmical until it reaches a maximum which is sustained at a fairly constant level during deep sleep.

On being awakened from sound sleep by command, the volume falls a to normal level, although at times there may be a brief rise before the fall in volume occurs. The pulse form continues high in amplitude with high prominent dicrotic wave for some times as long as 50 seconds after awakening and then with the fall in volume the characteristic pulse form of the waking state appears. (See Fig. 1, Plate I.)

All our record consistently gave sustained increased volume during sleep, but we had to introduce variations in our usual procedure to determine whether the rhythmical rise in volume precedes sleep or occurs after sleep ensues. This question is

very important in relation to the so-called 'cerebral congestion' theory of Stevenson et al. They, like Howell, believe that the sympathetic center in the brain controlling vasomotor tone becomes periodically fatigued, but, unlike Howell, believe it produces vaso-dilatation of the blood vessels of the brain sufficient to pull apart the neurones or alter the synapse and produce sleep. To make an explanation of sleep dependent upon a mechanical pulling apart of the neurones or alteration of synaptic connections, it is necessary to show that the supposedly fatigued sympathetic center produces the cerebral congestion before or as sleep ensues, and the congestion disappears before or as the subject awakens.

In our study we found it relatively easy to show that the subjects report being awake, after arousal from sound sleep, while the volume curve is still high with the characteristic pulse form of sleep; a situation that would be unlikely if contractility of neurones, or alteration of synapse from intracranial pressure, were the cause of sleep. It was very difficult on the other hand, to show that the increased volume did not precede the onset of sleep, because there are no adequate criteria to distinguish the moment sleep ensues, if there is such a moment. The observer made notations of when snoring appeared, but that proved an ineffective criterion. Critical examination of the respiratory tracing also proved of only little value.

Another method used in an attempt to answer this question was to have several of the subjects press a key until impairment of consciousness ensued. The rise in volume with sleep did not appear until after pressing the key had stopped, but this again is a poor criterion of the moment sleep occurs. Probably the best evidence that the circulation changes are the result rather than the cause of sleep was obtained on the occasions the subject was aroused just as the volume curve began to ascend in the characteristic fashion of sleep. The introspections on these occasions indicated that the subject had already fallen asleep. The subject could not be very positive that he or she had been truly asleep, but it was agreed that some impairment of

consciousness had already occurred. After due consideration that the criteria set up to determine the exact moment sleep ensues are not adequate, we do believe that the circulatory changes are the result rather than the cause of sleep.

The effect of the command "Wake up!" on the volume curve of sleeping subjects has already been discussed, but there are interesting differences produced by the same auditory stimulation given with the subjects awake that should be mentioned. Whereas in sleep the command arouses the subject and produces a diminution in volume with a pulse of smaller amplitude, the same command while awake produces a short temporary rise in volume without definitely changing the pulse form. It is interesting to observe that sensory stimulation such as determining of blood pressure, sound of pounding radiators, and other extraneous noises that occurred during the sleep observations, are liable to affect the circulation of blood during sleep even though they do not awaken the sleeping subject. The sensory stimulation evidently acts reflexly upon the vasomotor center, causing a contraction of the relaxed blood vessels. The resulting diminution, however, is never as great as when the subject is actually aroused. This is strikingly similar to observations made on arm volume during sleep by Howell and illustrated in his text book (9). Of further interest in this connection is that Loomis, Harvey, and Hobart, in measuring electrical potentials from the brain during sleep, report that here too auditory stimulation which is insufficient to awaken the sleeping subject, produces in some individuals momentary alpha waves that are characteristic of the waking state rather than that of sleep. The best evidence we have that there is a latent period in the contraction of relaxed vessels following auditory stimulation, is from our early experiments. At that time the kymograph motor, which was later repaired, started out with a loud sound and then continued very quietly. Since during sleep, when no changes were apparent in the writing lever, the motor was stopped for lines of reference and to conserve paper, it was possible to detect the temporal sequence of the loud sound of starting with the diminution in volume produced in sleep.

Blood pressure during sleep has been studied by other investigators and found to be lower in sleep than when awake at rest. During sleep we found a sustained increased volume of the blood in the brain, so we were interested in substantiating the lowering of the blood pressure in sleep. Subject 1 would awaken too quickly for blood pressure determinations, but we did get data from subject 2, sufficient to show that the blood pressure decreases a significant amount during deep sleep. The decrease in this subject was from 100 mm/60 mm Hg at rest awake, to an average of 86 mm/60 mm Hg during deep sleep.

The rise in blood pressure when awakened by command is abrupt and as already shown by other investigators may exceed that of the normal awake at rest period. This abrupt rise at awakening occurs while the brain vessels evidently are still relaxed, as shown by arterial pulse of high amplitude and prominent elevated dicrotic. It is conceivable that this sudden increase of blood pressure, occurring at a time when the brain vessels are still relaxed, may be a dangerous period to the individual already having a diseased condition of the cerebral arteries.

To determine the heart action during sleep the volume curve was studied in still another way. Rate of the heart beat was counted while awake at rest, and compared with a corresponding period during sleep. The heart rate, we found, was slightly slower in sleep than awake. Evidently, then, the rise in volume with sleep is accompanied by lowered heart rate and a slight decrease of blood pressure.

Our method of studying changes in the respiration while awake, sleeping, and during induced hypnosis, with the Sumner pneumograph placed over the thorax, is not very adequate in that we do not have records of abdominal breathing. If the breathing decreased during sleep, presumably there might be a slight accumulation of carbon dioxide content of the blood sufficient to act as a stimulus to produce relaxation of the cerebral vessels. Subject 2 demonstrated periodic breathing during sleep. With restricted breathing the volume increased slightly; with deep breathing the volume decreased.

However, in general, sleep in all but one subject showed increased rather than decreased respiration. In spite of this, the volume always remained high during sleep.

Subject 1 maintained a relatively regular respiration throughout sleep with the amplitude of the wave slightly decreased over that awake. However, experiments upon artificially increased breathing followed by shallow breathing while awake clearly showed that the decreased breathing does not explain the marked sustained rise of brain volume and the pulse form that appears in sleep. Restricted breathing awake causes a slight rise in volume without an apparent change of pulse form, whereas in sleep the volume increased markedly with changes in amplitude and form of the arterial pulse.

Results of Induced Hypnosis Observations

There are twenty records of induced hypnosis in two of our subjects. We have described the distinct changes that appear in the cerebral circulation with the onset of natural sleep. With the onset of hypnotic sleep the changes of cerebral circulation did not appear. The cerebral circulation during induced hypnosis resembled the waking state rather than that of sleep. The arterial pulse cannot be distinguished from that in a corresponding wake period in amplitude, or form of pulse wave.

When hypnosis is induced there is a temporary rise in volume without changing the amplitude of the arterial pulse. This, however, is similar to the reaction to all auditory stimuli given to the subject in the normal wake period.

Hypnosis on four different occasions merged into natural sleep with one subject. When this occurred, the brain volume showed the distinct changes found in sleep. We knew that natural sleep had occurred by testing for hypnosis. The subject awakened as soon as the arm was grasped preparatory to testing for catalepsy. This test we found would differentiate hypnosis from the waking state or natural sleep in this subject.

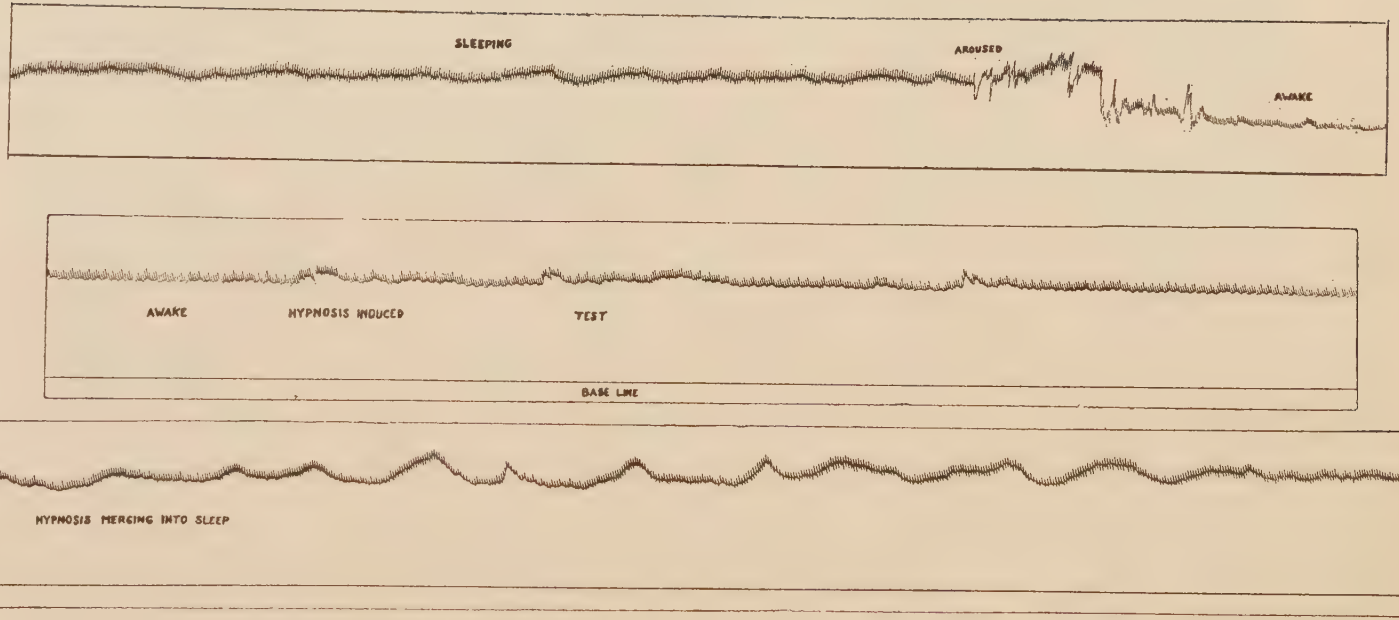
Description of Plates

The observations on cerebral circulation prevailing during sleep and hypnosis can best be illustrated by excerpts from typical records. Unfortunately, due to the expense, only a few of the records are herein published. They have been selected to show the characteristic differences between awake, trance, and sleep.

March 6, 1937. Plate I, Fig. 1. Subject No. 1

The subject was in bed in the laboratory with the apparatus connected and ready to record by 8:20 P.M. After a wait of 10 minutes to allow for temperature adjustment to take place, the lights were turned out in the sleeping room and the recording begun. While the subject was at rest but still awake the command, "Wake up!" was given. Similar awake reactions to the words "Wake up!" had been recorded on other evenings so that comparisons could be made with reactions in sleep. The response in the volume curve while awake was always a short brief rise with no change in amplitude of pulse or position of the dicrotic. The excerpt shown in Fig. 1 is from the second period of sleep. She had been asleep earlier, awakened by command, and allowed to fall asleep again. When sleep ensues, usually from 3 to 6 rhythmical rises are observed in this subject before the volume attains a maximum level which is sustained throughout deep sleep. This excerpt begins 17 minutes after full and sustained volume appeared in sleep. At the point marked 'aroused' the subject awakened quickly to the command, "Wake up!". In spite of a quick awakening, the volume remained sustained for 44 seconds after being aroused with the characteristic pulse form of sleep. Sudden, abrupt rises and falls of the record after 'aroused' are due to stretching tense muscles after a period of sound sleep. These movements are almost impossible to eliminate. In spite of this sharp break upward from stretching, all curves from this subject showed a fall in volume after awakening. Note, however, how slowly the constriction with awakening occurs.

PLATE I


SLEEPING
AROUSED
AWAKE
AWAKE
HYPNOSIS INDUCED
TEST
BASE LINE
HYPNOSIS MERGING INTO SLEEP

April 2, 1937. Plate I, Fig. 2. Subject No. 1

The experiment for this evening was designed as a continuation of the study of hypnosis. We had already obtained a record in which hypnosis merged into sleep in this subject and we were interested primarily in obtaining more of this type. The subject reported being very sleepy. The apparatus was all attached by 8:10 P.M. After a brief observation on the effect of changing from deep to shallow breathing while awake, hypnosis was induced, followed by a test for catalepsy. Near the end of the second kymograph paper of the evening, she was aroused by the same command as was used in normal sleep, namely, "Wake up!" Figure 2 of Plate I is an excerpt taken about 10:15 P.M. at the beginning of the third change of paper. She had been awake for about 10 minutes and the volume curve pointer was still writing at the same level and with similar pulse as at the end of the second paper. Note especially the amplitude of the pulse and the position of the dicrotic. Hypnosis suggestions began at the point where the volume curve ascends slightly. This momentary rise in volume without a change in pulse form is a characteristic response, in all curves obtained during wake or hypnotic periods, to all forms of auditory stimulation whether it be suggestions for hypnosis or the command, "Wake up!".

The second slight rise denotes where the arm was lifted to test for catalepsy; usually the test for catalepsy was made without verbal suggestions but in this instance I suggested: "Your arm is getting more and more rigid." When it attained full rigidity, a suggestion for general relaxation and deeper sleep was given while the arm was placed on the bed. The last portion of the record of Fig. 2 shows the typical form of the cranial volume curve at all times during induced hypnosis. Note that it cannot be distinguished from the pulse that is in evidence while awake. We hoped to let this pulse continue to see if it eventually merged into the characteristic volume change and pulse form of sleep. If and when the volume curve showed the characteristic rise and pulse form of sleep we were prepared to test for hypnosis and not before.

To test for the trance condition at regular intervals may act as a stimulus suggestion for hypnosis. We thought that this interval testing of hypnosis may account for our earlier records never merging into sleep in spite of lengthy records.

April 21, 1937. Plate I, Fig. 3. Subject 1

This 9-minute natural-size excerpt illustrates hypnosis merging into sleep. It was taken from the same record as Fig. 2 about 30 minutes later. During the interim no changes in pulse form or volume were observed. Incidentally, we never obtained sighing, stretching, or position changes during hypnosis, although these were sometimes evident when we studied comparative awake or asleep periods. The piston was still writing at the same level at the beginning of this excerpt as it had been at the end of the excerpt shown in Fig. 1. Sleep probably began just before the first rhythmical rise in the tracing. It took a good deal of study to show when trance or awake periods ended, and sleep ensued. This question, since it required separate experimentation, can be more profitably discussed elsewhere in this paper. Note in this excerpt the rhythmical manner the curve rises when sleep occurs. Accompanying the volume rise are the typical changes in pulse form that occur in sleep in this subject. Note the position of the dicrotic wave in deep sleep at the end of this excerpt. The amplitude of the pulse wave is greater than observed in trance states or wake periods and in addition the dicrotic notch becomes elevated and more pronounced.

Not shown is the sustained increased volume that followed this excerpt. It remained sustained until the operator grasped the arm of the sleeping subject to test for catalepsy as usual. This immediately awakened the subject, and the volume curve slowly descended to the normal level very similar to that shown in Fig. 1.

Introspections were often obtained after a subject had been aroused from either sleep or hypnosis. On one of these occasions she reported, "I think I know now how I can tell whether you awakened me from natural sleep or hypnotic sleep by estimating how sleepy I feel; when I am aroused

from hypnosis I am wide awake immediately, but when awakened from natural sleep, I still feel drowsy."

June 12, 1937. Plate II, Fig. 1. Subject No. 3

This subject is an epileptic boy with a left temporal decompression. We hoped to obtain records during an epileptic seizure but were unsuccessful. This excerpt shows the respiration and brain volume curve and illustrates: sleeping, being aroused, a short wake period, and drowsiness.

July 17, 1937. Plate II, Fig. 2. Subject 4

This excerpt illustrates a normal sleep record with an arousal produced by testing for catalepsy. Elevating the arm always aroused this subject with the same reaction in the volume curve as the command, "Wake up!".

July 19, 1937. Plate II, Fig. 3. Subject No. 4

This is an excerpt from a typical hypnosis session in subject no. 4. It begins with the subject awake, then hypnosis was induced followed by suggestion for localized analgesia in one arm. The short brief reflex rise in volume near the center of the excerpt is where the test for analgesia was made by piercing the skin with a pin. The facial expression did not indicate any feeling of pain. When the non-analgesic arm is pierced, the reflex rise in volume is similar, but the facial expression and bodily movements indicate pain. In spite of lengthy records, hypnosis in this subject never merged into natural sleep.

Relationship of Results to Circulatory Theories

The so-called 'cerebral anæmia' theory of sleep is found untenable as an explanation of sleep in our subjects. On the contrary, rather than a condition of cerebral anæmia, our observations indicate that with the onset of sleep the volume of blood in the brain increases slightly with a more pronounced pulse.

Our data supports the findings of Shepard and Stevenson *et al.*, that cerebral congestion is an accompaniment of sleep, but it does not agree with the cerebral congestion theory

PLATE II

SLEEPING
AROUSED
AWAKE
SLEEPING
TEST
AWAKE
HYPNOSIS INDUCED
TEST

of sleep as postulated by Stevenson *et al.* We have shown that the circulatory changes do not precede the onset of sleep; and that the circulation changes are therefore more likely to be the result of, rather then because of, sleep. We have also shown that the subjects report being awake after arousal while the volume curve is still high, a condition that would be unlikely if sleep is to be explained by mere mechanical pulling apart of the neurones from the increased intracranial pressure.

Conclusions

Four young men and one young woman, having recovered from accidents that necessitated removal of a portion of the skull, acted as subjects in the observation of cerebral circulation during sleep. In two of these subjects a profound somnambulistic hypnotic trance was induced. From twenty observations upon induced hypnosis from these subjects and thirty-four records of sleep from all we summarize our results as follows:

The volume of blood in the brain increases during sleep with a more pronounced pulse, which we envisage as being due to relaxed blood vessels. The rise in volume is gradual and rhythmical until it reaches a maximum which is sustained at a fairly constant level during deep sleep.

On being awakened, the volume curve descends to a normal level, although at times there may be a short rise before the volume fall occurs. Accompanying the circulation changes are the slower heart rate and slight decrease of blood pressure in sleep.

An auditory stimulus which is insufficient to awaken the sleeping subject is likely to produce a diminution in the volume of the brain; whereas, the same auditory stimulus while awake is likely to produce a rise in volume.

With the onset of hypnotic sleep the changes of cerebral circulation did not appear. The cerebral circulation prevailing during hypnosis resembles the waking state rather than that of sleep. The pulse cannot be distinguished from a corresponding wake period in amplitude, form of pulse wave, or volume.

Howell's so-called 'cerebral anæmia' theory of sleep and

the 'cerebral congestion' theory of Stevenson, Christianson, Wortis, the author considers inadequate as an explanation of sleep in his subjects. There are changes in the cerebral circulation with the onset of sleep but these changes are more likely the result of sleep rather than the cause. This author believes that we must look elsewhere than in our present circulation theories for an explanation of sleep.

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